



**Issue Date: 16 June 2004**

**IN THE MATTER OF:**

OLA MAE PRICE (widow of deceased  
miner William Price),  
Claimant,

v.

Case No.: 1997-BLA-1676

CONSOLIDATION COAL CO.,  
Employer,

**and**

DIRECTOR, OFFICE OF WORKERS'  
COMPENSATION PROGRAMS,  
Party-in-Interest.

**DECISION AND ORDER ON REMAND  
AWARDING SURVIVOR'S BENEFITS**

On March 19, 2003, the Benefits Review Board (Board) issued a *Decision and Order* vacating, in part, the *Order on Remand – Granting Benefits* dated January 4, 2000 by Administrative Law Judge John C. Holmes. The Board held the following:

- the ALJ must reconsider the chest x-ray evidence under 20 C.F.R. § 718.202(a)(1) to determine whether pneumoconiosis is established;
- the opinions of Drs. Zaldivar and Castle with regard to whether the miner suffered from pneumoconiosis were improperly discredited;
- it was error to find that Drs. Abernathy, Kress, Fino, Endres-Bercher, and Crisalli appeared to diagnose "legal pneumoconiosis" where they found an "obstructive lung disease" where, as noted by the Board, these physicians attributed the lung impairment to the miner's tobacco abuse;
- it was improper to discredit the opinions of Drs. Morgan and Zaldivar on the etiology of the miner's death where, although they did not diagnose the presence of pneumoconiosis, they "assumed" the presence of the disease for purposes of their reports.

- the ALJ properly accorded little weight to the opinion of Dr. Morgan with regard to the existence of pneumoconiosis;
- the ALJ properly credited the opinions of Drs. Ducatman, Buono, and Rasmussen that smoking and coal dust exposure “led to the miner’s fatal respiratory failure”;
- the ALJ properly accorded less weight to the opinions of Drs. Fino, Castle, and Endres-Bercher with regard to the cause of the miner’s death, but the Board stated the following:  
  
[B]ecause the administrative law judge’s credibility determinations as to the opinions of Employer’s experts on the cause of the miner’s death are dependent on his determination on the existence of pneumoconiosis at 20 C.F.R. § 718.202, a finding which we herein vacate, we also vacate the administrative law judge’s finding at 20 C.F.R. § 718.205(c).
- the ALJ is required to apply *Compton* and weigh all of the evidence under 20 C.F.R. § 718.202(a) together to determine whether pneumoconiosis is established;
- the ALJ’s findings of 42 years of coal mine employment and 35 to 45 pack years of smoking were affirmed; and
- the ALJ’s finding that pneumoconiosis, if present, was caused by coal dust exposure under 20 C.F.R. § 718.203 (2001) was affirmed.

By *Order* dated October 29, 2003, the undersigned Administrative Law Judge canceled the hearing scheduled on remand and the parties’ request for a decision on the record was granted. This *Decision* is based on the evidence admitted into the record as well as the arguments of the parties on remand.

## I Chest x-ray evidence under 20 C.F.R. § 718.202(a)(1) (2001)

The regulation at 20 C.F.R. § 718.202(a)(1) (2001) requires that “where two or more X-ray reports are in conflict, in evaluating such X-ray reports consideration shall be given to the radiological qualifications of the physicians interpreting such X-rays.”<sup>1</sup> In this vein, the Board has held that it is proper to accord greater weight to the interpretation of a B-reader or Board-certified radiologist over that of a physician without these specialized qualifications. *Roberts v. Bethlehem Mines Corp.*, 8 B.L.R. 1-211 (1985); *Allen v. Riley Hall Coal Co.*, 6 B.L.R. 1-376 (1983). Moreover, an interpretation by a dually-qualified B-reader and Board-certified

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<sup>1</sup> A “B-reader” (B) is a physician, but not necessarily a radiologist, who successfully completed an examination in interpreting x-ray studies conducted by, or on behalf of, the Appalachian Laboratory for Occupational Safety and Health (ALOSH). A designation of “Board-certified” (BCR) denotes a physician who has been certified in radiology or diagnostic roentgenology by the American Board of Radiology or the American Osteopathic Association. An “A-reader” is a physician, but not necessarily a radiologist, who submitted six x-ray studies of his or her clients to ALOSH of which two studies are interpreted as positive for the existence of pneumoconiosis, two studies are negative, and two studies demonstrate complicated pneumoconiosis.

radiologist may be accorded greater weight than that of a B-reader. *Roberts v. Bethlehem Mines Corp.*, 8 B.L.R. 1-211 (1985); *Sheckler v. Clinchfield Coal Co.*, 7 B.L.R. 1-128 (1984).

The undersigned incorporates and adopts the chart summarizing the chest x-ray evidence as set forth in ALJ Holmes' *Decision*.<sup>1</sup> Of the chest x-ray studies of record, two physicians conclude that the March 1982 study is negative for the presence of pneumoconiosis. One physician found that the study was unreadable. As a result, this study does not support a finding of pneumoconiosis.

Similarly, the June 1983, July 1983, January 1985, April 1985, August 1985, and August 1986 studies produced negative readings for the presence of pneumoconiosis.

A January 1987 study was interpreted as positive by four dually-qualified physicians, whereas 1 dually-qualified physician and two B-readers found insufficient evidence of the disease. On balance, a preponderance of the more qualified physicians interpreted the study as positive and this study supports a finding of pneumoconiosis.

Two studies dated September 1987 were interpreted as positive by two dually-qualified physicians. On the other hand, four dually-qualified physicians and three B-readers concluded that the study was negative. A preponderance of the dually-qualified physicians, as well as three B-readers, submitted negative interpretations such that this study does not support a finding of the disease.

The January 1988, June 1989, April 1991, October 1993, March 1995, and June 1995 studies produced negative findings.

A July 1995 study yielded positive interpretations by four dually-qualified readers, whereas only two dually-qualified physicians found insufficient evidence of the disease. This study supports a finding of pneumoconiosis.<sup>2</sup>

One of two August 2, 1995 studies was unreadable. Another study conducted on the same date was interpreted negatively by two dually-qualified readers and a reader with unknown qualifications. These studies do not support a finding of pneumoconiosis. Similarly, a March 1996 study produced negative findings.

A January 3, 1997 study, although read by one radiologist as demonstrating "no active cardiopulmonary disease," and a B-reader determined that it was negative, two dually-qualified readers concluded that the study was unreadable such that it is not probative of the presence or absence of pneumoconiosis.

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<sup>1</sup> The chart is modified to reflect that Dr. Morgan (KWM) is a B-reader. Moreover, there is evidence of record that the following physicians are dually-qualified: Drs. Wiot, Felson, Spitz, Bassali, Speiden, DeRamos, Cappiello, Aycoth, Shipley, and Ahmed. It is noted that Dr. Pathak is a B-reader and is board-certified in radiology in Britain.

<sup>2</sup> ALJ Holmes' chart of the chest x-ray evidence is modified to reflect that the July 25, 1997 study interpreted as negative by Dr. Shipley on December 26, 1997 should have been dated as a July 25, 1995 study.

A January 8, 1997 study did not yield positive findings of pneumoconiosis. The last study of record, dated July 1997, was interpreted by a dually-qualified reader as negative for the presence of pneumoconiosis and, thus, also does not support a finding of the disease.

On balance, only the January 1987 and July 1995 studies can be considered as positive for the presence of pneumoconiosis. The remaining readable studies of record, including the most recent study of record, produced negative results for the presence of pneumoconiosis according to dually-qualified physicians. Consequently, Claimant has not sustained her burden of establishing pneumoconiosis based on the chest x-ray evidence of record under 20 C.F.R. § 718.202(a)(1) (2001).

## II

### **Medical opinion evidence under 20 C.F.R. § 718.202(a)(4) (2001)**

The second method by which Claimant in this case may establish that the miner suffered from the disease is by well-reasoned, well-documented medical reports. A “documented” opinion is one that sets forth the clinical findings, observations, facts and other data on which the physician based the diagnosis. *Fields v. Island Creek Coal Co.*, 10 B.L.R. 1-19 (1987). An opinion may be adequately documented if it is based on items such as a physical examination, symptoms, and the patient’s history. *See Hoffman v. B&G Construction Co.*, 8 B.L.R. 1-65 (1985); *Hess v. Clinchfield Coal Co.*, 7 B.L.R. 1-295 (1984).

A “reasoned” opinion is one in which the administrative law judge finds the underlying documentation adequate to support the physician’s conclusions. *Fields, supra*. Indeed, whether a medical report is sufficiently documented and reasoned is for the administrative law judge as the finder-of-fact to decide. *Clark v. Karst-Robbins Coal Co.*, 12 B.L.R. 1-149 (1989)(en banc). Moreover, statutory pneumoconiosis is established by well-reasoned medical reports which support a finding that the miner’s pulmonary or respiratory condition is significantly related to or substantially aggravated by coal dust exposure. *Wilburn v. Director, OWCP*, 11 B.L.R. 1-135 (1988).

All of the examining and consultative physicians of record conclude that the miner suffered from severe, progressively worsening chronic obstructive lung disease. Drs. Rasmussen, Buddington, Cardona, Moore, Mitchell, Hynes, Ducatman, Buono, and Hatahet concluded that the miner’s respiratory impairment was due, at least in part, to his coal dust exposure as well as to tobacco abuse. The remaining physicians, Drs. Abernathy, Kress, Fino, Endres-Bercher, Crisalli, Castle, and Zaldivar, conclude that the miner did not suffer from coal workers’ pneumoconiosis and his respiratory impairment was due to his tobacco abuse. Some of these physicians also noted an “asthmatic component” to the miner’s respiratory condition. Physical examinations of the miner consistently revealed the presence of sounds in the lungs, including wheezing, distant breath sounds, or rhonchi.

#### A. Dr. Robert Abernathy

Dr. Abernathy examined and tested the miner and issued a report on August 13, 1986. He reported a history of 41 years of coal mine employment, where the miner continued to work

as a dispatch operator, and a 40 pack year history of cigarette smoking, quitting in 1985. Examination of the lungs revealed “audible expiratory wheezes throughout both sides” and distant lung sounds “in all areas. Dr. Abernathy noted that the lung sounds “were very difficult to hear. He further observed that the miner’s chest “appeared to be somewhat hyperinflated with some elevation of the sternum” and the “AP diameter was moderately increased.”

Dr. Abernathy diagnosed the miner with severe chronic obstructive pulmonary disease with reversible bronchospasm as well as chronic bronchitis and probable cor pulmonale. He concluded that there was “no indication of coal workers’ pneumoconiosis.” Dr. Abernathy concluded that the miner was “totally incapacitated from any type of work except for that of dispatching, which requires no exertion at all.” He further states the following:

The question is the cause of the pulmonary insufficiency and whether significant smoking history would appear (sic) that this is the predominant factor in producing his respiratory problems. The studies would be consistent with advanced coal workers’ pneumoconiosis, as seen in progressive massive fibrosis but since he obviously does not have this, the coal workers’ pneumoconiosis may be excluded as the basic cause of his problem. Exposure to coal dust probably contributed to the chronic bronchitis when he was exposed to coal dust, but since he has been removed from high levels of coal dust, it would not appear that the coal dust is causing his chronic bronchitis at this time. The chronic bronchitis at this time is a result of his emphysema, however.

Dr. Abernathy noted that a chest x-ray was negative for the presence of pneumoconiosis. Blood gas testing revealed impaired diffusion and deterioration on exercise. Pulmonary function testing demonstrated obstruction with reversible bronchospasm.

On May 21, 1988, Dr. Abernathy issued a supplemental letter to respond to certain questions by Employer. He stated that the miner suffered from a respiratory impairment caused by his 45 pack year history of cigarette smoking. Based on medical data available to him, Dr. Abernathy concluded that the miner did not suffer from coal workers’ pneumoconiosis.

Conclusions. Dr. Abernathy’s opinions regarding the existence of coal workers’ pneumoconiosis are entitled to little probative value. While his finding of no clinical pneumoconiosis is supported by the preponderantly negative chest x-ray interpretations of record, the only form of legal coal workers’ pneumoconiosis addressed by Dr. Abernathy is chronic bronchitis. In particular, he found that, at one time, the miner’s chronic bronchitis was probably due to his coal dust exposure. However, Dr. Abernathy concluded that “it would not appear that coal dust (was) causing” the miner’s bronchitis at the time of his examination as the miner ceased working by that point.

On the other hand, Dr. Abernathy concludes that emphysema was causing the miner’s post-employment bronchitis. He does not explain why 42 years of coal mine employment did not also contribute to the miner’s respiratory impairment (*i.e.* bronchitis caused by emphysema). In *Cannelton Industries, Inc. v. Director, OWCP [Frye]*, Case No. 03-1232 (4<sup>th</sup> Cir. Apr. 5,

2004)(unpub.)<sup>3</sup>, the Fourth Circuit recently concluded that a similar opinion was entitled to little probative value. The court noted that Dr. Forehand found the miner to be totally disabled due to smoking-induced chronic bronchitis but failed to explain “how he eliminated (the miner’s) nearly thirty years of exposure to coal mine dust as a possible cause” of the bronchitis. The court concluded that “Dr. Forehand erred by assuming that the negative x-rays (underlying his opinion) necessarily ruled out that (the miner’s) bronchitis was caused by coal mine dust . . .” In this case, Dr. Abernathy’s opinions are similarly not well-reasoned or well-documented and the undersigned is not persuaded by Dr. Abernathy’s conclusions.

B. Dr. George O. Kress

Dr. Kress conducted a review of certain medical records and submitted a report on February 17, 1988. He noted a 39 year history of coal mine employment as well as a 45 pack year cigarette smoking history. Dr. Kress agreed with Dr. Fino’s analysis of the medical evidence and concluded that there was insufficient objective evidence to justify a diagnosis of coal workers’ pneumoconiosis based on preponderantly negative chest x-ray evidence. Moreover, Dr. Kress opined that the miner suffered from a “marked degree of pulmonary impairment” and he diagnosed the presence of advanced chronic obstructive pulmonary disease, chronic bronchitis and pulmonary emphysema. Dr. Kress cites to certain medical literature for the proposition that, while coal dust exposure may cause chronic bronchitis in some miners, “this is usually not of a severe degree” and that severe chronic bronchitis is usually caused by cigarette smoking. As a result, he concludes that the miner’s respiratory conditions are due to his tobacco abuse and he opined that the miner would “continue to experience further deterioration in his lung function as his emphysema progresses.” Further, Dr. Kress states that, even if the miner suffered from the presence of “very early pneumoconiosis not visible by x-rays”, such a condition would not “cause pulmonary impairment of an obstructive type, nor would it, in any way cause hypoxemia demonstrated in his blood gas studies.”

Conclusions. Dr. Kress’ opinion that the miner suffered from obstructive lung disease arising only from smoking is not well-reasoned. His citation to negative chest x-ray interpretations addresses only the clinical form of the disease.

Moreover, citation to various articles, which purportedly stand for the proposition that smoking, and not coal dust exposure, causes obstructive lung disease is not persuasive. To the contrary, the Fourth Circuit and comments to the Department of Labor’s amendments to the regulations provide that coal workers’ pneumoconiosis may cause purely obstructive lung disease. 20 C.F.R. § 718.201(a)(2) (2001); *Warth v. Southern Ohio Coal Co.*, 60 F.3d 173 (4<sup>th</sup> Cir. 1995). As a result, Dr. Kress’ apparent disagreement with this premise entitles his report to less probative weight. *See also Freeman United Coal Mining Co. v. Summers*, 272 F.3d 473 (7<sup>th</sup> Cir. 2001) (ALJ properly accorded less weight to a physician’s opinion that coal dust inhalation not cause significant obstructive lung disease where Department dispensed with similar presentation during rulemaking proceedings for amended regulations). Moreover, Dr. Kress dismisses coal dust exposure as contributing to the miner’s chronic bronchitis stating that such exposure “usually” does not in any severe form of bronchitis. He did not explain this opinion in

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<sup>3</sup> A copy of the Fourth Circuit’s unpublished decision is attached to this opinion.

light of the miner's specific symptoms and smoking and work histories. *Knizer v. Bethlehem Mines Corp.*, 8 B.L.R. 1-5 (1985) (a medical opinion based on generalities, rather than specifically focusing on the miner's condition, may be accorded less weight). In essence, Dr. Kress' opinion that the miner's progressively deteriorating obstructive lung disease was due solely to tobacco abuse, with no persuasive explanation excluding contribution of the miner's 42 years of exposure to coal dust, carries little probative value. *See Frye, supra*.

C. Dr. Gregory Fino

Findings. Dr. Fino's earliest consultative report in this case is dated October 22, 1987. He noted a 39 year history of coal mine employment and a 45 pack year cigarette smoking history. Based on the medical data available to him, Dr. Fino concluded that the miner suffered from a moderately severe obstructive lung disease, which rendered him totally disabled. He opined, however, that the miner's lung disease was not due to coal dust exposure because (1) the chest x-ray evidence was preponderantly negative, (2) cigarette smoking is the most common cause of large airways disease (obstruction) and it is "unusual" for coal workers' pneumoconiosis to cause such a disease, (3) pulmonary function testing reveals a response to bronchodilators, which is inconsistent with coal workers' pneumoconiosis, (4) medical literature supports a finding that the miner's decreasing FEV1 values are most consistent with chronic obstructive pulmonary disease secondary to cigarette smoking, (5) the miner's blood gas testing produced variable results which is not consistent with coal workers' pneumoconiosis, (6) the miner's lung condition has improved with treatment, which is not consistent with the presence of coal workers' pneumoconiosis, and (7) there is no "good, statistically significant evidence to show that industrial bronchitis causes pulmonary disability."

By report dated October 8, 1993, Dr. Fino cited to particular articles in support of his opinion that the miner did not suffer from coal workers' pneumoconiosis. Specifically, Dr. Fino referenced Dr. Francis Green's 1992 presentation in the *American Review of Respiratory Disease*:

(Dr. Green) found that the most common form of emphysema in smokers who were not coal miners was centriacinar emphysema. In coal miners with and without cigarette smoking, focal emphysema was found. Clearly, Dr. Green is showing that there is a difference between focal emphysema and centriacinar emphysema.

Dr. Fino opined that focal emphysema is due to coal dust exposure and is asymptomatic whereas centrilobular emphysema is the "dominant form of emphysema in smokers." Based on certain articles, Dr. Fino concluded that "[t]here is no evidence of an increase in disabling or impairing emphysema in coal miners. Although emphysema is a pathological entity seen in coal workers' pneumoconiosis, coal workers' pneumoconiosis does not cause clinical emphysema."

In his most recent report dated November 11, 1997, Dr. Fino agreed with Dr. Rasmussen that the miner "died a respiratory death due to significant chronic obstructive lung disease." However, Dr. Fino noted that the miner's "significant hypercarbia" was determinative of the

etiology of the miner's lung disease. In particular, he opined that hypercarbia, which is when a person's carbon dioxide level exceeds 45, "has never been described as a part of coal workers' pneumoconiosis when the chest x-ray readings are negative or even as high as 1/1" as in this case.

Dr. Fino noted that the miner experienced significant progression of his lung disease since the late 1980s and the miner developed hypercarbia over the next ten years:

Coal workers' pneumoconiosis is not going to progress in the absence of further coal mine dust exposure unless there is . . . significant pulmonary fibrosis such as a Category 3 x-ray or complicated pneumoconiosis. However, cigarette smoking induced lung disease is known to cause this type of worsening of lung function over time even if a person stops smoking.

Finally, Dr. Fino asserts that "[t]he medical literature clearly shows an accelerated reduction in lung function in individuals who have smoked, even if they quit smoking," which is "consistent with the clinical course as recorded in the record."

During his December 4, 1997 deposition, Dr. Fino testified that, even the few positive chest x-ray readings of record "do not show enough scarring and fibrosis to produce an obstructive abnormality that would be related to coal mine dust inhalation. *Ex. 7* at 11. Moreover, blood gas testing over time revealed "progressive elevation in the carbon dioxide levels, which is a "classic picture of progressive obstructive lung disease secondary to cigarette smoking." Dr. Fino further opined the following:

In order to get an elevation in the carbon dioxide level, there has to be very significant lung destruction present. Very significant lung destruction due to coal workers' pneumoconiosis is the type of lung destruction that's obvious on the chest x-ray.

*Ex. 7* at 11.

Dr. Fino explained that "hypercarbia" is when the carbon dioxide level in the miner's bloodstream exceeds the normal of 45. *Ex. 7* at 15. Dr. Fino stated that, if he assumed the presence of coal workers' pneumoconiosis, then he would find that it contributed to the miner's death. *Ex. 7* at 18. He noted that only miner's with industrial bronchitis will suffer from a purely obstructive impairment but a "mild to minor obstruction goes away 6 to 12 months after a miner leaves the mines." *Ex. 7* at 19.

Conclusions. Dr. Fino's opinions are not persuasive for three reasons. First, he states that coal workers' pneumoconiosis cannot develop in the absence of continued exposure unless a miner suffers from a complicated form of the disease or, at a minimum, has a chest x-ray reading of Category 3 or greater. The amended regulations at 20 C.F.R. § 718.201 (2001) provide that pneumoconiosis is recognized as a "latent and progressive disease which may first become detectable only after the cessation of coal mine dust exposure." *National Mining Ass'n. et al v. Director, OWCP [Kramer]*, 305 F.3d 203 (3<sup>rd</sup> Cir. 2002) (upholding regulation as valid). *See*

also *Lane Hollow Coal Co. v. Lockhart*, 137 F.3d 799, 803 (4<sup>th</sup> Cir. 1992) (pneumoconiosis is progressive and irreversible). There is no threshold requirement that a miner produces a chest x-ray reading of Category 3 or suffer from complicated pneumoconiosis prior to finding that the disease is progressive. Indeed, in *Barnes v. Mathews*, 562 F.2d 278, 279 (4<sup>th</sup> Cir. 1977), the court held that “pneumoconiosis is a slow, progressive disease often difficult to diagnose at early stages . . .” In its comments to the amended regulations, the Department considered Dr. Fino’s position and found that “[c]ontrary to Dr. Fino’s conclusions, a number of medical references document the latent progressive nature of the disease.” 65 Fed. Reg. 79,970 (Dec. 20, 2000). The Department then cited to several medical articles demonstrating that simple, as well as complicated, pneumoconiosis may be latent and progressive.

Second, Dr. Fino posits that hypercarbia, which is when a person’s carbon dioxide level exceeds 45, “has never been described as a part of coal workers’ pneumoconiosis when the chest x-ray readings are negative or even as high as 1/1” as in this case. Dr. Fino’s summary conclusion is not persuasive. *Cosaltar v. Mathies Coal Co.*, 6 B.L.R. 1-1182 (1984) (a physician’s report may be accorded less weight where the basis for the opinion cannot be determined). He does not explain why hypercarbia cannot be caused by Category 1 coal workers’ pneumoconiosis or a form of legal pneumoconiosis that is not readily discernible on chest x-rays. Indeed, since Dr. Fino agrees that the miner suffered from chronic obstructive pulmonary disease and centrilobular emphysema, it is logical that one or both conditions contributed to the development of the miner’s hypercarbia and, as will be discussed, the undersigned finds that coal dust exposure contributed to these conditions.

Third, Dr. Fino opined that the miner suffered from centrilobular emphysema arising from tobacco abuse. He stated that, while coal dust exposure causes focal emphysema, this form of emphysema does not manifest symptoms. Dr. Rasmussen cited to an NIOSH article titled “Criteria for a Recommended Standard Occupational Exposure to Respirable Coal Mine Dust,” to state that smoking and coal dust exposure can cause centrilobular emphysema. As will be discussed, the undersigned finds that Dr. Rasmussen’s citation to the NIOSH article, combined with Dr. Fino’s failure to adequately explain why coal dust exposure does not contribute to the development of centrilobular emphysema, requires that less probative weight be accorded Dr. Fino’s opinion in this regard. Moreover, in its comments to the amended regulations, the Department takes a position contrary to Dr. Fino in this regard:

Drs. Fino and Bahl find no scientific support that clinically significant emphysema exists in coal miners without progressive massive fibrosis . . . , but the available pathologic evidence is to the contrary.

. . .

Centrilobular emphysema (the predominant type observed) was significantly more common among the coal workers. The severity of the emphysema was related to the amount of dust in the lungs. These findings held even after controlling for age and smoking habits.

65 Fed. Reg. 79,942 (Dec. 20, 2000). Notably, the Seventh Circuit, in *Freeman United Coal Mining Co. v. Summers*, 272 F.3d 473 (7<sup>th</sup> Cir. 2001), accorded less weight to Dr. Fino's opinion where it contradicted the findings of the Department:

Dr. Fino stated in his written report . . . that 'there is no good clinical evidence in the medical literature that coal dust inhalation in and of itself causes significant obstructive lung disease.' (citation omitted). During a rulemaking proceeding, the Department of Labor considered a similar presentation by Dr. Fino and concluded that his opinions 'are not in accord with the prevailing view of the medical community or the substantial weight of the medical and scientific literature.

Consequently, Dr. Fino's opinion is entitled to less weight in this case because it is neither well-reasoned nor well-documented and his opinion is premised on findings contrary to established case law as well as the Department's position in its comments to the amended regulations.

D. Dr. Gregory Endres-Bercher

Dr. Endres-Bercher examined and tested the miner and issued a report on September 9, 1987. He noted a 40 year coal mine employment history as well as a 44 pack year history of tobacco exposure. Examination of the miner revealed that his breathing was "slightly labored" with audible wheezing as well as inspiratory and expiratory rhonchi. The miner's chest was hyperexpanded with "hyperresonance on percussion." A chest x-ray was interpreted as normal. Pulmonary function testing revealed a severe obstructive pulmonary disease and blood gas testing produced evidence of "mild hypoxemia at rest." Dr. Endres-Bercher concluded that the miner suffered from severe obstructive lung disease and emphysema secondary to the miner's tobacco abuse. He noted the following:

Obstructive lung disease is not characteristic of coal workers' pneumoconiosis. The chest x-ray does not show changes consistent with an interstitial process. In addition, coal workers' pneumoconiosis shows a restrictive pattern on spirometry which is not present on today's exam.

By supplemental report dated November 18, 1997, Dr. Endres-Bercher opined that air trapping is a hallmark of obstructive lung disease. He noted that the miner exhibited an increase in PCO<sub>2</sub> values due to air trapping. Dr. Endres-Bercher further opined that coal workers' pneumoconiosis is a restrictive lung disease and he stated the following:

Since all of (the miner's) pulmonary function testing is repeatedly consistent with the presence of chronic obstructive pulmonary disease, I would not consider coal workers' pneumoconiosis a contributing factor to Mr. Price's disability or death even if confirmed by x-ray findings."

Dr. Endres-Bercher noted that the miner never demonstrated restrictive defects on testing.

Conclusions. The undersigned Administrative Law Judge does not find that Dr. Endres-Bercher's opinion is well-reasoned or well-documented. In particular, Dr. Endres-Bercher appears to rule out the presence of coal workers' pneumoconiosis on grounds that the miner demonstrated an obstructive, as opposed to restrictive, impairment. As noted in conjunction with the opinions of certain other physicians, the amended regulations and Fourth Circuit acknowledge that legal coal workers' pneumoconiosis may manifest itself as a purely obstructive impairment. 20 C.F.R. § 718.201(a)(2) (2001); *Warth, supra*; *Summers, supra*. Moreover, while negative chest x-ray readings preclude the presence of clinical pneumoconiosis, they do not document a finding of no legal pneumoconiosis. As with certain other physicians of record, Dr. Endres-Bercher fails to explain how 42 years of coal dust exposure did not contribute to the miner's progressive respiratory disease. *See Frye, supra*. While the undersigned notes that extensive coal mine employment does not compel a finding that such employment contributed to the development of a miner's respiratory disease, it is incumbent upon the medical experts to provide an adequate explanation regarding causation. *Fuller v. Gibraltar Corp.*, 6 B.L.R. 1-1292 (1984) (an unsupported medical conclusion is not a reasoned diagnoses). Moreover, as with Dr. Fino's opinion, Dr. Endres-Bercher's report is premised on a finding that simple coal workers' pneumoconiosis does not produce an obstructive impairment, which is contrary to the weight of medical literature cited in support of the Department's contrary position in its comments to the amended regulations. *See Summers, supra*.

E. Dr. Robert J. Crisalli

By report dated October 30, 1991, Dr. Crisalli conducted examinations of the miner on April 4 and 23, 1991 and conducted a review of certain medical records. Dr. Crisalli reported 44 years of coal mine employment as well as a 30 to 35 pack year history of smoking cigarettes, where the miner quit smoking six to seven years prior to the examination. Examination of the miner revealed an increased expiratory phase and wheezing consistent with chronic bronchitis and emphysema. Moreover, the miner exhibited diminished chest wall motion, which also indicated the presence of emphysema. A chest x-ray produced negative findings and pulmonary function testing demonstrated a "severe obstruction" with bronchodilator response. Dr. Crisalli noted that the miner's carboxyhemoglobin level was "borderline" elevated. He issued a conclusory statement that the miner did not suffer from coal workers' pneumoconiosis, but that he suffered from tobacco induced chronic bronchitis and emphysema.

Conclusions. Dr. Crisalli's unreasoned opinion is not probative. Although Dr. Crisalli's testing and examination support his finding of chronic bronchitis and emphysema, his conclusory statement regarding the etiology of these conditions is unpersuasive. *Duke v. Director, OWCP*, 6 B.L.R. 1-673 (1983) (a report is properly accorded less probative value where the physician does not explain how underlying documentation supports his or her diagnosis). As previously noted, negative chest x-ray results are insufficient to document the absence of legal coal workers' pneumoconiosis.

F. Dr. James Castle

In his November 1997 report, Dr. Castle stated, without explanation, that he observed no physical findings that would indicate the presence of coal workers' pneumoconiosis. He further stated that radiographic evidence did not support a finding of the disease.

Pulmonary function testing demonstrated a moderately severe obstructive airways disease, which progressed from 1987 until the miner's death, which Dr. Castle found was consistent with tobacco smoke induced pulmonary emphysema and chronic bronchitis. Dr. Castle also noted that the miner suffered from gas trapping and a reduction in diffusing capacity, which were consistent with pulmonary emphysema.

Dr. Castle noted a significant reversibility of the miner's obstruction, but he failed to mention the presence of asthma noted by several other physicians of record.

Dr. Castle opined that "[w]hen coal workers' pneumoconiosis causes clinically significant impairment, it does so by causing a mixed irreversible obstructive and restrictive ventilatory process." Moreover, he stated that, if coal workers' pneumoconiosis was significant enough to cause gas trapping, then "very significant changes" above a Category 1 would be noted in the chest x-rays, which was not the case here.

Dr. Castle noted that the miner's blood gas study results indicated a "frank respiratory insufficiency" with an elevation in the PCO<sub>2</sub>. He noted that coal workers' pneumoconiosis causes persistent hypoxemia without causing a CO<sub>2</sub> elevation. Dr. Castle concluded that the miner's medical data was "absolutely typical of respiratory failure associated with tobacco smoke induced pulmonary emphysema."

Dr. Castle testified, during his December 1997 deposition, that the miner had evidence of a "moderately severe degree of obstructive airways disease which (he) felt had progressed from about 1987 until his demise." *Ex. 6 at 12.* Dr. Castle found no coal workers' pneumoconiosis based on a majority of chest x-ray readings. *Ex. 6 at 14.* He noted the following:

As might be expected in an individual with tobacco smoke-induced pulmonary emphysema, (the miner) developed a progressive respiratory insufficiency such that he had now developed progressive hypoxemia requiring oxygen therapy, and then the finding of what we call hypercarbia, or increased PCO<sub>2</sub>.

That is – those are hallmarks of respiratory failure due to tobacco smoke-induced pulmonary emphysema, and are not associated with the findings of coal workers' pneumoconiosis.

*Ex. 6 at 15-16.* After noting that the miner last worked as a dispatcher and left the mines in 1985, Dr. Castle further stated the following:

The hallmarks of this process are that it was progressive after he left the mines with no further coal dust exposure. He had progressive hypoxemia and CO<sub>2</sub>

retention, which is one of the hallmarks of respiratory failure from tobacco smoking.

*Ex. 6* at 17. Dr. Castle maintained that the miner did not exhibit a mixed impairment, which further militated against a finding of coal workers' pneumoconiosis. *Ex. 6* at 20.

*Conclusions.* Dr. Castle's opinions are unpersuasive for four reasons. First, Dr. Castle stated, without explanation, that he did not observe any consistent physical findings that would indicate the presence of pneumoconiosis; rather, he concluded that the miner's symptoms were consistent with smoking-induced chronic obstructive pulmonary disease and emphysema. In this vein, he failed to address the miner's consistent symptoms of shortness of breath, dyspnea, wheezing, distant breath sounds, or rhonchi noted on multiple examinations. *Mabe v. Bishop Coal Co.*, 9 B.L.R. 1-67 (1986) (an unsupported medical conclusion is not a reasoned diagnosis). While such symptoms are not necessarily diagnostic of the presence of coal workers' pneumoconiosis, it is incumbent on the medical experts to provide a well-reasoned diagnosis in light of the available medical data. Dr. Castle's failure to address why these symptoms are solely attributable to the miner's smoking history, given the extent of his coal mining and smoking histories, detracts from the persuasiveness of his report.

Second, Dr. Castle fails to address certain discrepancies and inconsistencies between the medical data and conclusions in his reports. For example, Dr. Castle notes that the miner's obstruction is reversible to some extent which, on its face, tends to militate against a finding of coal workers' pneumoconiosis. However, he also noted that the miner's overall respiratory condition progressively worsened from 1987 until his death.

By recent decision in *Consolidation Coal Co. v. Director, OWCP [Swiger]*, Case No. 03-1971 (4<sup>th</sup> Cir. May 11, 2004)(unpub.)<sup>4</sup>, the Fourth Circuit held that partial reversibility did not preclude the presence of coal workers' pneumoconiosis where the miner was left with a "residual fully disabling impairment." The *Swiger* court stated the following:

The evidence shows that when Swiger was given bronchodilator medication, his pulmonary condition improved, but the residual impairment that remained was still disabling. All the experts agree that pneumoconiosis is a fixed condition and therefore any lung impairment caused by coal dust would not be susceptible to bronchodilator therapy. In this case, although Swiger's condition improved when given a bronchodilator, the fact that he experienced a disabling residual impairment suggested that a combination of factors was causing his pulmonary condition. As a trier of fact, the ALJ 'must evaluate the evidence, weigh it, and draw his own conclusions.' (citations omitted). Therefore, the ALJ could rightfully conclude that the presence of the residual fully disabling impairment suggested that coal mine dust was a contributing cause of Swiger's condition. (citation omitted).

In this case, Dr. Castle reviewed Dr. Zaldivar's medical records wherein it was noted that the miner suffered from asthma. Indeed, Dr. Zaldivar noted that reversibility in the miner's lung

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<sup>4</sup> A copy of the Fourth Circuit's unpublished opinion is attached to this decision.

obstruction was attributable to asthma and Dr. Castle stated that Dr. Zaldivar found “improvement after bronchodilator is significant *only in so far as the percentage but not as far as the absolute improvement.*” (emphasis added). Dr. Castle does not adequately address the underlying data in this regard and his conclusions regarding the data are not well-reasoned.

As previously noted, the physicians of record agree that the miner’s overall respiratory condition progressively worsened and his response to a bronchodilator was not significant in terms of his overall lung function over time. The fact that the miner had a totally disabling residual impairment lends further support to a finding that he suffered from coal workers’ pneumoconiosis.

Third, Dr. Castle reported that Dr. Zaldivar found that the miner was a current smoker based on his “significant” carboxyhemoglobin level. However, when Dr. Zaldivar’s underlying data is reviewed, the carboxyhemoglobin level was 2.0 which, according to Dr. Zaldivar, was “borderline” between a smoker and non-smoker. This data does not support a finding that the miner continued to smoke. Thus, characterizing the carboxyhemoglobin level as “significant” and that the miner was a current smoker is not supported by data underlying his report or the report of Dr. Zaldivar. This also renders Dr. Castle’s opinion regarding the existence of coal workers’ pneumoconiosis less reliable. *See Trumbo v. Reading Anthracite Co.*, 17 B.L.R. 1-85 (1993) (a physician’s opinion is less probative where based on inaccurate smoking history).

Fourth, Dr. Castle noted that the miner suffered from hypercarbia, which he asserts is a “hallmark of respiratory failure due to tobacco smoke-induced pulmonary emphysema” and it is “not associated with findings of coal workers’ pneumoconiosis.” Dr. Castle fails to explain his conclusion and he does not provide documentation to support his statement that hypercarbia is “not associated with findings of coal workers’ pneumoconiosis.” Indeed, his opinion is inconsistent with that of Dr. Fino, who states that findings of Category 1 pneumoconiosis or less precludes pneumoconiosis as a cause of hypercarbia. Dr. Castle offers a more sweeping statement that hypercarbia is not associated with coal workers’ pneumoconiosis, apparently regardless of the extent of the disease. Moreover, Dr. Castle does not persuasively explain why hypercarbia is caused only by smoking-induced emphysema and could not also be the result of legal coal workers’ pneumoconiosis (which was not visible on chest x-ray studies). As a result, Dr. Castle’s opinion is not probative on this ground. *See Cosaltar, supra.*

#### G. Dr. George Zaldivar

Dr. Zaldivar agreed that the miner died due to severe obstructive lung disease. He concluded that this disease arose from tobacco abuse and asthma. Dr. Zaldivar noted that there was insufficient objective data to support a finding of coal workers’ pneumoconiosis. He found the presence of a “severe irreversible airway obstruction with air trapping and a moderate diffusion impairment all of which were due to (the miner’s) smoking habit.”

After his October 22, 1991 examination, Dr. Zaldivar opined that the miner suffered from smoking-induced emphysema with an asthmatic component. He found no evidence of coal workers’ pneumoconiosis by chest x-ray. Dr. Zaldivar further concluded that Dr. Rasmussen’s

opinion was incorrectly based on a finding of no air trapping; rather, Dr. Zaldivar stated that his testing revealed significant air trapping. Dr. Zaldivar noted that the miner had a high carboxyhemoglobin level compatible with a current smoking habit. This conclusion is not supported by the data underlying his report as Dr. Zaldivar noted that the miner's carboxyhemoglobin level at 2.0 is "borderline" between a smoker and a non-smoker.

Dr. Zaldivar issued a supplemental report on January 7, 1998 based on a review of certain medical records. He noted severe, irreversible airway obstruction with air trapping and a moderate diffusing impairment due to the miner's smoking habit. Dr. Zaldivar reported that the miner continued to smoke at the time of his 1991 examination "as evidenced by the high carboxyhemoglobin level measured in (his) office." Dr. Zaldivar noted some improvement after use of a bronchodilator due to the asthmatic component of the miner's disease. He concluded that there was insufficient evidence to justify a diagnosis of coal workers' pneumoconiosis. Dr. Zaldivar opined that the miner suffered from a very significant pulmonary impairment due to smoking-induced emphysema.

Conclusions. Initially, Dr. Zaldivar stated that the miner's carboxyhemoglobin level was "compatible with a current smoking habit." However, he acknowledged that the recorded level was 2.0, which was "borderline" between the level expected of a non-smoker and a smoker. The preponderance of the evidence of record demonstrates that the miner quit smoking in 1986. While Dr. Zaldivar notes that the miner was a smoker in 1991, the underlying data and other testimony of record indicates that the miner was a non-smoker at that time. Dr. Zaldivar's failure to accurately address the medical data regarding the miner's smoking history, and his unsupported conclusion that the miner had a current "significant" smoking habit, detracts from the reliability of his report. *See Trumbo, supra.*

Second, Dr. Zaldivar's finding of no coal workers' pneumoconiosis appears to be based on negative chest x-ray findings and the fact that pulmonary function testing revealed an obstructive impairment without restriction. As previously noted, a finding of no clinical pneumoconiosis based on negative chest x-rays is consistent with the undersigned's determination in this case. However, negative chest x-rays do not preclude a finding of legal pneumoconiosis. Moreover, the amended regulations and Fourth Circuit acknowledge that legal coal workers' pneumoconiosis may manifest itself as a purely obstructive impairment. 20 C.F.R. § 718.201(a)(2) (2001); *Warth, supra*; *Summers, supra*. In this vein, Dr. Zaldivar's does not adequately explain why only smoking contributed to the miner's obstructive impairment, or why the miner did not suffer from legal coal workers' pneumoconiosis. *See also Frye, supra* (the Fourth Circuit upheld an ALJ's rejection of similar opinions that the miner did not suffer from coal workers' pneumoconiosis based on negative chest x-ray and obstructive, partially reversible findings on pulmonary function testing).

#### H. Drs. Alan Ducatman and Gina Buono

Drs. Ducatman and Buono reviewed certain medical records and issued a joint report on October 21, 1997. They reported 35 to 40 years of coal mine employment as well as a 35 to 45 pack year history of smoking cigarettes, where the miner quit in 1986. In particular, they

reviewed hospitalization records and a series of medical reports by Drs. Rasmussen, Mitchell, and Gaziano along with the underlying chest x-ray, blood gas testing, and pulmonary function testing. Drs. Ducatman and Buono noted that the miner had “progressive effort dyspnea for the past 12-15 years” and he suffered from “episodes of coughing, paroxysmal nocturnal dyspnea and ankle swelling” as well as “wheezing and decreased breath sounds.” Blood gas testing revealed “reduced arterial oxygen and a diminished diffusing capacity.” Drs. Ducatman and Buono Dr. Rasmussen’s exercise test “showed marked impairment in gas exchange with hypoxia, indicating some pulmonary insufficiency.”

Drs. Ducatman and Buono diagnosed the presence of clinical and legal pneumoconiosis. They concluded that severe chronic obstructive pulmonary disease, arising from cigarette smoking and coal workers’ pneumoconiosis, was the immediate cause of the miner’s death. They also found the presence of simple coal workers’ pneumoconiosis based on a single positive chest x-ray interpretation by a B-reader. Drs. Ducatman and Buono further diagnosed the presence of right ventricular heart failure secondary to severe lung disease, and coronary artery disease caused by cor pulmonale and cigarette smoking.

Conclusions. Drs. Ducatman and Buono concluded that the miner suffered from coal workers’ pneumoconiosis based on a positive chest x-ray interpretation. They also found that the miner suffered from chronic obstructive pulmonary disease arising from coal dust exposure and tobacco abuse and that this condition caused his death. As with certain other medical reports of record, a finding of *clinical* coal workers’ pneumoconiosis is not supported by a preponderance of the chest x-ray evidence of record, which detracts from the persuasiveness of their opinion. However, these physicians also found the presence of *legal* pneumoconiosis, *i.e.* chronic obstructive lung disease arising from smoking and coal dust exposure. This diagnosis is supported by a preponderance of the evidence of record, including the miner’s symptoms and blood gas and pulmonary function testing results. Drs. Ducatman and Buono noted accurate smoking and coal mine employment histories and their report is based on a review of certain medical records. As a result, the undersigned finds that this report supports a finding of *legal* coal workers’ pneumoconiosis.

Employer argues on remand that the opinion of Drs. Ducatman and Buono is not probative since these physicians did not review examination or consultative reports from any physicians concluding that the miner did not suffer from coal workers’ pneumoconiosis. The Board has already disagreed with this argument. (*See Board’s Decision* at 13). As previously noted, the report of Drs. Ducatman and Buono is based on testing, symptoms, and accurate smoking and coal mine employment histories. As such, their report is well-documented and well-reasoned.

#### I. Dr. Richard S. Buddington

Dr. Buddington examined the miner and issued a report on April 19, 1985. He noted a 45 pack year history of smoking cigarettes and a 40 year history of coal mine employment. Based on pulmonary function and blood gas testing as well as physical examination, Dr. Buddington noted that the miner suffered from a moderate, chronic respiratory impairment. He attributed the

miner's respiratory impairment to coal dust exposure based on "many years of mining." He did not offer any further explanation of his diagnosis. After reviewing a positive x-ray reading by Dr. Bassali, Dr. Buddington reiterated his diagnosis of coal workers' pneumoconiosis in a letter dated February 20, 1987.

Conclusions. Dr. Buddington's opinion supports a finding of coal workers' pneumoconiosis. It is noteworthy that he diagnosed presence of the disease based on symptoms and blood gas and pulmonary function testing, prior to receiving Dr. Bassali's positive x-ray interpretation. Therefore, while Dr. Buddington's finding of clinical pneumoconiosis is not tenable on this record, his finding of legal pneumoconiosis is supported by the medical data underlying his report. *See Fields, supra.*

J. Dr. Larry G. Mitchell

Dr. Mitchell examined and tested the miner and issued a report on September 18, 1989. He noted that the miner suffered from worsening dyspnea, cough, and congestion. Dr. Mitchell reported a 45 pack year cigarette smoking history and that the miner had a history of pneumoconiosis per Dr. Abernathy. Examination of the lungs revealed wheezing, dry rales at the bases, and distant breath sounds. Dr. Mitchell also noted a "barreling" of the chest. A chest x-ray revealed hyperinflation consistent with chronic obstructive pulmonary disease. Dr. Mitchell noted that the chest x-ray was not read for diagnosing black lung. Pulmonary function testing demonstrated severe obstructive airways disease. Dr. Mitchell diagnosed the presence of chronic obstructive pulmonary disease and history of black lung. He noted that the miner's lung disease was progressively worsening and that his overall prognosis was poor.

Conclusions. Dr. Mitchell's opinion does not support a finding of coal workers' pneumoconiosis. Specifically, Dr. Mitchell appears to assume the presence of the disease based on Dr. Abernathy's report. Dr. Mitchell's examination of the miner revealed symptoms that may be consistent with coal workers' pneumoconiosis, but Dr. Mitchell did not provide any independent reasoning with regard to the issue. As a result, his report is not well-reasoned. *See Fuller, supra.*

K. Hospitalization and treatment records

Dr. E.E. Moore issued a report on September 5, 1985 stating that he had treated the miner since February 1981, when the miner was admitted to the hospital for "chronic obstructive pulmonary disease and acute bronchitis." Upon Dr. Moore's last examination of the miner on July 29, 1985, he noted "high-pitched wheezing" and diminished breath sounds as well as an increased AP diameter in the chest. He diagnosed the miner with severe chronic obstructive pulmonary disease secondary to his history of coal dust exposure. Dr. Moore noted that the miner was a smoker, but he did not specify the extent of the miner's smoking history.

Dr. Moore issued a supplemental report on March 18, 1987 after reviewing certain medical records. He reiterated that the miner suffered from advanced chronic obstructive

pulmonary disease secondary to coal workers' pneumoconiosis based on numerous chest x-ray opinions and a long history of underground coal mining. Dr. Moore opined that the miner was totally disabled due to the disease.

Dr. Mario Cardona treated the miner during certain hospitalizations. On July 25, 1995, Dr. Cardoza prepared a hospital admission report for shortness of breath, cough, and chest pain. Dr. Cardoza reported at least 30 years of coal mine employment and that the miner was not a smoker. Examination revealed that the miner's AP diameter was increased and he had a barrel chest. Moreover, there was audible wheezing and rales in all the pulmonary fields. He diagnosed the miner with acute respiratory disease and acute exacerbation of chronic obstructive pulmonary disease. Dr. Cardoza also found that the miner suffered from arteriosclerotic heart disease and impending congestive heart failure. In his discharge report dated August 18, 1995, Dr. Cardoza added the diagnosis of "black lung."

By letters dated February 25, 1987, June 16, 1988, July 3, 1989, and May 27, 1994, Dr. Cardona repeatedly concluded that the miner suffered from a totally disabling respiratory impairment arising, at least in part, from coal dust exposure. Although in his June 28, 1989 hospital admission report Dr. Cardoza noted that the miner "quit smoking many years ago," he failed to specify the extent of this smoking history. Moreover, the record supports a finding that the miner quit smoking around 1986.

Dr. Yasir Hatahet served as the miner's consulting respiratory physician during his January 1997 hospitalization. Dr. Hatahet diagnosed the miner with respiratory distress due to severe airflow obstruction. During his December 1997 deposition, Dr. Hatahet testified that he was board-certified in internal medicine. He treated the miner during his January 1997 hospitalization and noted that the miner was in respiratory distress and had a low oxygen level and obstructive airway disease. He stated that, based on his recollection of the chest x-ray evidence, the miner had "extensive interstitial fibrosis" and coal workers' pneumoconiosis. He stated the following:

[S]omebody that has coal workers' lung disease, and they come in with pneumonia, they're not going to cope with it as well as somebody who has no underlying or pre-existing lung condition, because most or at least some of their functional lung tissue is gone by the pre-existing abnormality.

Blood gas testing revealed a "severe abnormality with gas exchange" and pulmonary function studies demonstrated "progressive impairment of lung function." Dr. Hatahet testified that coal dust exposure usually causes restriction, but "coal dust disease can by itself without any other etiology cause airway obstruction, as well." Even if he assumed a 30 pack year smoking history, Dr. Hatahet would find that, while smoking contributed to the miner's chronic obstructive lung disease, the miner still had chest x-ray evidence of coal workers' pneumoconiosis.

Dr. A. Hynes treated the miner during his January 1997 hospitalization. He noted in the admission report that the miner suffered from imminent respiratory failure, chronic obstructive pulmonary disease with exacerbation, black lung, and atherosclerotic heart disease. Dr. Hynes reported a 30 year history of coal mine employment, but did not note a smoking history.

Examination of the lungs revealed diffuse wheezing, poor air excursion, and bronchovesicular breath sounds. He diagnosed the miner with impending respiratory failure, chronic obstructive pulmonary disease with exacerbation, black lung, and theophylline toxicity.

Dr. R.K. Krishnan conducted a pulmonary consultation for the miner during his January 1997 hospitalization. Dr. Krishnan noted that the miner “never smoked” and that he worked in the coal mines for 44 years. He stated that the miner is “known to have COPD complicated by (coal workers’ pneumoconiosis).” Dr. Krishnan stated that the miner suffered from a chronic respiratory insufficiency and that he was “oxygen dependent at home.” Examination in the emergency room revealed respiratory distress, severe coughing, and wheezing. There was “diminished air exchange in all lung fields,” inspiratory and expiratory rhonchi, wheezing, and coarse rales.

During his December 1997 deposition, Dr. Krishnan testified that he is board-certified in internal medicine, geriatrics, pulmonary medicine, and critical care. He has practiced pulmonary medicine in Bluefield, West Virginia since 1973 and served as the miner’s emergency room doctor on January 4, 1997. Dr. Krishnan recalled that, on admission, the miner was in “marked respiratory distress” and was “smothering.” Dr. Krishnan stated that the miner was gasping for breath and was coughing. He noted a 44 year history of coal mine employment and stated that, to his knowledge, the miner never smoked. Dr. Krishnan diagnosed the miner with chronic obstructive pulmonary disease and emphysema arising from coal dust exposure.

Conclusions. The miner’s treating physicians opine that he suffered from a respiratory impairment arising, in part, from coal dust exposure. Standing alone, however, this evidence would be insufficient to sustain Claimant’s burden given the fact that the physicians were vague regarding the miner’s smoking history, or they stated that he never smoked. *See Trumbo, supra*. Moreover, certain treating physicians based a finding of coal workers’ pneumoconiosis on positive chest x-ray evidence, which is not supported by a preponderance of the x-ray evidence of record. *Scott v. Mason Coal Co.*, 289 F.3d 263 (4<sup>th</sup> Cir. 2002) (ALJ may accord less weight to an opinion that is based on a premise contrary to the ALJ’s findings). Consequently, the hospitalization and treatment records support a finding that the miner suffered from coal workers’ pneumoconiosis, but they are not sufficient, standing alone, to sustain Claimant’s burden in this regard.

L. Dr. D.L. Rasmussen

Dr. Rasmussen examined and tested the miner and issued a report on January 20, 1987. He noted a 41 and one-half year history of coal mine employment as well as a 35 pack year cigarette smoking history, where the miner quit one year ago. Blood gas testing demonstrated “markedly” reduced resting values as well as a “markedly reduced” diffusing capacity. The miner’s EKG was normal and Dr. Rasmussen concluded that the miner suffered from totally disabling respiratory disease due to tobacco abuse and coal dust exposure.

Based on his February 4, 1987 examination, Dr. Rasmussen diagnosed the presence of coal workers’ pneumoconiosis based on a chest x-ray and prolonged exposure to coal dust. In

February 1993, after conducting a medical record review, Dr. Rasmussen concluded that the miner suffered from severe pulmonary emphysema related to cigarette smoking and coal dust exposure. He stated that the miner's lung disease "must be considered secondary to his occupational exposure, at least in major part in view of his prolonged exposure and X-ray evidence of pneumoconiosis."

By supplemental report dated August 22, 1989, Dr. Rasmussen stated that the miner suffered from occupational lung disease, which contributed to his overall totally disabling respiratory impairment. Dr. Rasmussen based his conclusion on the miner's normal lung volume, severe airway obstruction, and marked reduction in diffusing capacity.

In a supplemental report dated February 4, 1993, Dr. Rasmussen reviewed certain medical records and stated the following:

There is general agreement that the patient has disabling pulmonary insufficiency. The principal physiologic abnormality appears to be that of severe pulmonary emphysema.

Noting that the chest x-ray evidence produced conflicting interpretations, Dr. Rasmussen cited to a study of x-rays and autopsy evidence available on 100 miners. The study revealed that 20 percent with severe macular pneumoconiosis, 30 percent with a moderate grade of macular pneumoconiosis, and 29 percent of individuals with micronodular pneumoconiosis had x-rays interpreted by three B-readers as negative for the presence of the disease. Dr. Rasmussen posits that a negative chest x-ray interpretation did not preclude the presence of pneumoconiosis in the miner's lungs. Moreover, Dr. Rasmussen stated that Drs. Zaldivar and Crisalli "ignored" a growing body of medical evidence that chronic obstructive pulmonary disease and emphysema can be caused by coal dust exposure, even in the absence of chest x-ray evidence. Indeed, he maintains that centrilobular and focal emphysema can arise from coal dust exposure.

In his September 22, 1997 report, Dr. Rasmussen concluded that the miner developed a progressively severe respiratory insufficiency requiring multiple hospitalizations. He noted a 40 year history of coal mine employment and an unspecified smoking history.

Dr. Rasmussen diagnosed coal workers' pneumoconiosis based on positive chest x-rays and the miner's lengthy coal mine employment history.

The miner died on January 8, 1997 as a consequence of respiratory failure. Dr. Rasmussen noted that cigarette smoking is a well-known cause of chronic obstructive pulmonary disease, including emphysema. However, he also stated that coal mine dust exposure can cause chronic obstructive pulmonary disease, including centrilobular emphysema. He cited to articles and studies, including a September 1995 National Institute of Occupational Safety and Health (NIOSH) article titled "Criteria for a Recommended Standard Occupational Exposure to Respirable Coal Mine Dust," that smoking and coal dust exposure can cause centrilobular emphysema. Dr. Rasmussen concluded that coal workers' pneumoconiosis was a "material contributing factor" to the miner's death.

Conclusions. Dr. Rasmussen's series of reports are well-reasoned and well-documented. He notes the miner's smoking and coal dust exposure histories and his opinion is based on examination and testing of the miner, a review of certain medical records, and review of medical literature with regard to diagnosing coal workers' pneumoconiosis. Although Dr. Rasmussen's diagnosis of clinical pneumoconiosis is not supported by a preponderance of the x-ray evidence of record, his finding of legal pneumoconiosis is well-reasoned and well-documented. This opinion is based on a physical examination, testing, and a review of certain medical records over time. As a result, Dr. Rasmussen's opinion is considered to encompass the most complete view of the miner's health and is based on more extensive findings than other reports of record. *Church v. Eastern Associated Coal Corporation*, 20 B.L.R. 1-51 (1997).<sup>5</sup> In this case, Dr. Rasmussen's finding of legal coal workers' pneumoconiosis is supported by the testing and observations noted in his report.

Indeed, Dr. Rasmussen's diagnoses of chronic obstructive lung disease and centrilobular emphysema are consistent with the findings of a preponderance of the physicians of record. The fact that he attributes the development of these conditions to the miner's extensive coal mining and smoking histories is reasonable. Studies cited by Dr. Rasmussen that coal dust exposure as well as tobacco abuse causes centrilobular emphysema are persuasive and consistent with the Department's position in its comments to the amended regulations. *See Bethenergy Mines, Inc. v. Director, OWCP [Rowan]*, Case No. 01-2148 (4<sup>th</sup> Cir. Sept. 4, 2002) (unpub.) (the court upheld the undersigned's finding that centrilobular emphysema was caused by the miner's smoking and coal dust exposure based on literature cited by Dr. Rasmussen); *Summers, supra*.<sup>6</sup>

Finally, a finding of legal coal workers' pneumoconiosis is consistent with the fact that the miner's respiratory impairment has progressively worsened over time. *See Lane, supra*; *Swiger, supra*. In sum, Dr. Rasmussen's opinion is well-reasoned and well-documented. It is further supported by the findings and opinions of Drs. Ducatman, Buono, Buddington, and Mitchell as well as the numerous treatment and hospitalization records in this case.

### III Weighing evidence together under *Compton*

In *Island Creek Coal Co. v. Compton*, 211 F.3d 203 (4<sup>th</sup> Cir. 2000), the court held that, in order to establish pneumoconiosis, all evidence submitted under 20 C.F.R. § 718.202 (2001) must be weighed together. Specifically, the undersigned is required to compare chest x-ray findings under § 718.202(a)(1) with medical opinion findings under § 718.202(a)(4) to determine whether Claimant has sustained her burden.

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<sup>5</sup> Employer incorrectly asserts in its remand brief that Dr. Rasmussen's opinion is "based largely, if not completely, upon the chest x-ray evidence . . ." (*Employer's brief* at 18). This is an accurate statement only with regard to Dr. Rasmussen's original February 1987 report. His subsequent reports clearly set forth separate bases for a finding of legal coal workers' pneumoconiosis. Moreover, Employer incorrectly states that Dr. Rasmussen is not a B-reader. (*Employer's Brief* at 20). To the contrary, a review of the record establishes that he is a B-reader.

<sup>6</sup> A copy of the Fourth Circuit's unpublished decision is attached to this opinion.

In this particular claim, the undersigned Administrative Law Judge found no clinical pneumoconiosis present as the preponderance of the chest x-ray evidence was negative for presence of the disease. However, based on Dr. Rasmussen's reports, as supported by the medical data and reports of certain other physicians of record, the undersigned is persuaded that the miner suffered from legal coal workers' pneumoconiosis. All of the physicians agree that the miner had chronic obstructive pulmonary disease and emphysema. Dr. Rasmussen reasonably attributes these conditions to the miner's extensive coal mine employment and smoking histories. His opinion is based on testing, symptoms, observations, and certain medical literature, including NIOSH's September 1995 article finding that coal dust exposure, along with tobacco abuse, causes centrilobular emphysema. In its comments to the amended regulations, the Department stated that "NIOSH is the government agency charged with conducting research into occupationally-related health problems" and serves as the "statutory advisor to the black lung benefits program . . . ." 65 Fed. Reg. 79,939 and 79,951 (Dec. 20, 2000). In this vein, the Department noted that NIOSH studies demonstrate that a diagnosis of chronic obstructive pulmonary disease "includes disease processes characterized by airway dysfunction: chronic bronchitis, emphysema and asthma." 65 Fed. Reg. 79,939 (Dec. 20, 2000). Moreover, there is a body of scientific studies finding that "[d]eath from pneumoconiosis, chronic bronchitis, and emphysema has been related to cumulative dust exposure." 65 Fed. Reg. 79,951 (Dec. 20, 2000).

A determination of whether a miner's respiratory disease arose from his coal dust exposure must be based on the expert medical evidence in each claim, and it is incumbent upon the medical experts to provide well-reasoned, well-documented opinions. The miner had a 42 year history of coal mine employment and ceased working in 1985. He had a 35 to 45 pack year history of smoking cigarettes and quit in 1986. Both of these potential causative factors are extensive. Therefore, opinions based on a premise that simple coal workers' pneumoconiosis generally does not cause obstructive lung disease, emphysema, or chronic bronchitis are not considered persuasive. More explanation is required based on the miner's specific circumstances, particularly in light of the fact that the miner's respiratory impairment progressively worsened over time; he demonstrated consistent symptoms of wheezing, prolonged inspiratory or expiratory phases, and/or rhonchi on multiple examinations; he suffered from deficits in his oxygen transfer on blood gas testing; and he had significant coal mining and tobacco abuse histories. Indeed, the miner's overall respiratory condition worsened significantly over time even considering his response to bronchodilators due to the asthmatic component of his impairment. As previously noted, Drs. Castle and Zaldivar noted that "improvement after bronchodilator (was) significant only in so far as the percentage but not as far as the absolute improvement."

The physicians' emphasis on negative chest x-ray evidence and obstructive nature of the miner's impairment indicates that they are focused on *clinical*, rather than *legal*, pneumoconiosis. In *Swiger, supra*, the Fourth Circuit found similar opinions lacking in probative value:

Four out of the five physicians whom the ALJ discredited concluded that Swiger did not have pneumoconiosis because his impairment was obstructive in nature, despite the fact that legal pneumoconiosis may consist of an obstructive impairment. For example, Dr. Renn says that coal dust exposure would not cause

an ‘obstructive ventilatory defect.’ (citation omitted). Dr. Rosenburger says that ‘clinically significant obstructive lung disease . . . is not associated with coal mine dust exposure’; . . . Dr. Fino says that Swiger’s symptoms demonstrate ‘obstructive lung disease . . . [which] is not a pattern consistent with the contraction of lung tissue due to fibrosis as would be expected in simple coal workers’ pneumoconiosis.

. . .

These comments support the ALJ’s findings that the employer’s physicians were overwhelmingly focused on clinical rather than legal pneumoconiosis.

*See also Cornett v. Benham Coal Co.*, 227 F.3d 569 (6<sup>th</sup> Cir. 2000).

Based on the foregoing, the opinions of Drs. Abernathy, Kress, Fino, Endres-Bercher, Crisalli, Castle, and Zaldivar are less probative with regard to whether the miner suffered from legal pneumoconiosis to the extent they focused on negative chest x-rays and the obstructive nature of his impairment in rendering their opinions.<sup>7</sup>

Dr. Rasmussen’s opinion, on the other hand, is based on grounds consistent with the Department’s position, *i.e.* that coal dust exposure can cause obstructive lung disease, including centrilobular emphysema, and simple pneumoconiosis may progressively worsen even in the absence of continued exposure to coal dust. He properly takes into account the miner’s symptoms, test results, and the extensive work and smoking histories established on this record. Dr. Rasmussen reasonably concludes that smoking and coal dust exposure contributed to the miner’s severe, progressive obstructive lung disease. Considering the multitude of factors underlying his opinion, preponderantly negative chest x-ray findings do not detract from Dr. Rasmussen’s findings of legal coal workers’ pneumoconiosis.

After weighing all of the evidence under 20 C.F.R. § 718.202(a)(1) and (a)(4) (2001), it is determined that Claimant has established that the miner suffered from legal coal workers’ pneumoconiosis.

#### **IV Cause of death**

Of the physicians who addressed the cause of the miner’s death, the Board held that ALJ Holmes properly credited the opinions of Drs. Ducatman, Buono, and Rasmussen that “smoking and coal dust exposure led to the miner’s total respiratory failure.” Moreover, Dr. Hynes, one of the miner’s treating physicians, concluded that he died due to “black lung.” Drs. Fino, Castle, and Endres-Bercher, on the other hand, agreed that the miner died as a result of respiratory failure, but they attributed his respiratory impairment only to tobacco abuse. These physicians failed to diagnose either clinical or legal pneumoconiosis. This is contrary to the aforesaid

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<sup>7</sup> Dr. Morgan’s opinion regarding the existence of pneumoconiosis has not been discussed as the Board upheld ALJ Holmes’ decision to accord it little weight.

findings based on the weight of the evidence of record and, consequently, their opinions are less probative in determining the cause of the miner's death. *Scott v. Mason Coal Co.*, 289 F.3d 263 (4<sup>th</sup> Cir. 2002) (less weight may be accorded to physicians' opinions premised on a finding of no pneumoconiosis that is contrary to the ALJ's findings).

Drs. Zaldivar and Morgan also failed to diagnose the presence of clinical or legal pneumoconiosis. However, they stated that, even if the miner suffered from the disease, his death was due solely to smoking-induced obstructive lung disease. Specifically, Dr. Zaldivar stated the following:

Mr. Price had bullae radiographically. Bullae due to emphysema is never a manifestation of coal workers' pneumoconiosis. It is a manifestation of emphysema which causes centrilobular and, in the case of bullae, panacinar emphysema.

Similarly, Dr. Morgan stated that the miner's respiratory impairment was related to emphysema and small airways disease and that any minor presence of coal workers' pneumoconiosis would "not affect his lung function." He reiterated that there was no evidence that the miner suffered from coal workers' pneumoconiosis "based on radiographic examination." Dr. Morgan did conclude, however, that the miner's death was due to severe chronic airflow limitation (emphysema and chronic bronchitis) due to cigarette smoking.

Reliance solely on chest x-ray findings to determine the cause of death is not persuasive given the plethora of other medical data available in this record. For reasons previously set forth throughout this opinion, it is concluded that coal dust exposure, as well as tobacco abuse, caused development of centrilobular emphysema in the miner's lungs. Because Drs. Zaldivar and Morgan conclude that emphysema contributed to the miner's death, and the record in this case supports a finding that the miner's emphysema was due in part to coal dust exposure, their opinions do not detract from a finding that coal dust exposure contributed to the miner's death.

Even though Drs. Morgan and Zaldivar did not diagnose the presence of pneumoconiosis, they stated that, even if the disease was established, their opinions as to the cause of the miner's death would not change. Their reports are not well-reasoned and carry little probative value. In *Soubik v. Director, OWCP*, \_\_\_ F.3d \_\_\_, Case No. 03-1668 (3<sup>rd</sup> Cir. Apr. 30, 2004), the court held that a physician's failure to diagnose pneumoconiosis would have an adverse effect on his ability to assess whether the miner's death was due to the disease:

Common sense suggests that it is unusually exceedingly difficult for a doctor to properly assess the contribution, if any, of pneumoconiosis to a miner's death if he/she does not believe it was present.

Similarly, in this case, Drs. Zaldivar and Morgan did not believe that the disease was present. As a result, they offered only conclusory statements regarding the cause of the miner's death in the event that presence of pneumoconiosis was assumed. As a result, their opinions are not well-reasoned or well-documented in support of a finding that coal workers' pneumoconiosis did not contribute to the miner's death.

Consequently, a preponderance of the evidence supports a finding that the miner suffered from legal coal workers' pneumoconiosis and, based on the opinions of Drs. Ducatman, Buono, and Rasmussen, this impairment hastened his death.<sup>8</sup> 20 C.F.R. § 718.205 (2001).

### **ORDER**

IT IS ORDERED that Employer shall pay to Claimant, Ola Mae Price, all benefits to which she is entitled commencing as of January 1997, the month in which the miner died; and

IT IS FURTHER ORDERED that, within thirty days of receipt of this *Decision*, Claimant's counsel shall file, with this Office and with opposing counsel, a petition for a representatives' fees and costs in accordance with the regulatory requirements set forth at 20 C.F.R. § 725.366 (2001). Counsel for the Director and for Employer shall file any objections with this Office and with Claimant's counsel within twenty days of receipt of Claimant's counsel's petition. It is requested that the petition for services and costs clearly state (1) counsel's hourly rate and supporting argument or documentation therefor, (2) a clear itemization of the complexity and type of services rendered, and (3) that the petition contains a request for payment for services rendered and costs incurred before this Office only as the undersigned does not have authority to adjudicate fee petitions for work performed before the district director or appellate tribunals. *Ilkewicz v. Director, OWCP*, 4 B.L.R. 1-400 (1982).

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Thomas M. Burke

Associate Chief Administrative Law Judge

**NOTICE OF APPEAL RIGHTS:** Pursuant to 20 C.F.R. § 725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board within 30 (thirty) days from the date of this Decision by filing a Notice of Appeal with the Benefits Review Board at P.O. Box 37601, Washington, D.C. 20013-7601. A copy of this Notice of Appeal must also be served on Donald S. Shire, Associate Solicitor for Black Lung Benefits, 200 Constitution Avenue,

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<sup>8</sup> Employer challenges the weight accorded these physicians' opinions in its remand brief. (*Employer's brief* at 25-26). However, the Board has already upheld ALJ Holmes' decision to accord greater weight to these opinions that the miner's death was due, in part, to *legal* coal workers' pneumoconiosis. *Brinkley v. Peabody Coal Co.*, 14 B.L.R. 1-147 (1990) (application of law of the case principle).